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Functional studies of the kidney have afforded many striking possibilities and many interesting problems. The subject is so new, the methods so exact, and the interpretation of results so little understood, that the subject is an ideal one for research.

During the past year, I[1] had an opportunity to study with Dr. Frothingham the different nitrogenous diets in chronic nephritis from a functional point of view. In these cases, we found, as has been demonstrated by Widal[2] and others, that certain types of chronic nephritis were unable to excrete salt normally. In many cases, 10 gm. of sodium chlorid, when added to the diet, was excreted poorly, or not at all.

Bunge,[3] several years ago, in observations on animals and normal individuals, found that the increased intake of potassium salts caused an increased sodium salt excretion, and vice versa. This observation suggested to us the possibility of causing an increased sodium salt excretion, by the addition of potassium salt to the diet, in cases of nephritis in which it had been proved that there was a decreased ability of the kidney to excrete salt.

Selected cases with chronic nephritis were studied in the wards of the hospital. The methods used and the data recorded are exactly the same as those used in the paper[1] previously referred to. In this problem, each case was first studied functionally, and classified. In certain cases, when it had been proved that there was definite inability of the kidney to excrete 10 gm. of added sodium chlorid, the patient was given potassium chlorid. The salt was administered in a single dose of 5 or 10 gm., in 150 c.c. of water at 10 a. m.

In some of the cases there was an apparent increase of the salt excretion following the increased potassium chlorid intake; in other cases no apparent effect was produced.

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1. Frothingham, C., and Smillie, W. G.: A Study of Different Nitrogenous Diets in Chronic Nephritis, The Archives Int. Med., 1915, xv, 204.

2. Widal: Mouvement Med., 1913, i, 1.

3. Bunge: Ztschr. f. Biol., 1873, ix, 104.

Four typical tables will be given.

Case 1 (1170).—This was classified clinically as of chronic nephritis, arteriosclerosis and hypertension. Functionally, there was moderate inability to excrete salt, with practically normal nitrogen excretion. On the 17th, 5 gm. of potassium chlorid were given. No increase of salt excretion occurred. No ill effects were produced. Results are given in Table 1.

TABLE 1.—Results of Test in Case 1 (1170)

Date	Fluid Intake c.c.	Urine		Nitrogen		Chlorid		Mg. N per 100 c.c. Blood	Per Cent. Phthalein in 2 Hrs.	Blood Pressure
		24° Am. c.c.	Sp. Gr.	Intake Gm.	Output Gm.	Intake Gm.	Output Gm.			
May 9	.....	1,880	1.016	11.2	12.6	4.0	14.0	34.2	50	230-130
May 10	.....	745	1.022	11.2	10.6	4.0	3.2			
May 11	.....	890	1.021	11.2	12.4	4.0 NaCl 10 gm.	3.4			
May 12	.....	1,515	1.020	11.2	12.2	14.0	9.5			
May 13	.....	1,530	1.014	11.2	12.2	4.0	5.1			
May 14	.....	1,530	1.015	11.2 Urea 20 gm. 21.2	16.4	4.0	3.0			
May 15	.....	1,160	1.017	11.2	12.8	4.0	1.5			
May 16	.....	1,340	1.017	11.2	14.7	4.0	3.6	.....	59	
May 17	700	1,060	1.016	11.2	10.6	KCl 5 9.0	3.5	28.5		
May 18	1,310	840	1.020	11.2	10.3	4.0	2.8			
May 19	1,480	980	1.020	11.2	11.5	4.0	2.8			

Case 2 (1072).—This was classified clinically as of chronic nephritis with hypertension. Functionally there was marked inability to excrete salt, and moderate inability to excrete nitrogen. On May 5, 5 gm. of potassium chlorid were given, which was followed by an immediate increase in the chlorid output. No ill effects were produced. Results are given in Table 2.

TABLE 2.—Results of Test in Case 2 (1072)

Date	Fluid Intake c.c.	Urine		Nitrogen		Chlorid		Mg. N per 100 c.c. Blood	Per Cent. Phthalein	Blood Pressure
		24° Am. c.c.	Sp. Gr.	Intake Gm.	Output Gm.	Intake Gm.	Output Gm.			
April 16	1,100	1,480	1.015	10.7	3.1	4.0	7.2	.....	.....	190-100
April 17	1,300	1,100	1.010	9.7	3.0	3.0	1.2	31.8	60	
April 18	950	840	1.015	10.7	6.3	NaCl 10 gm. 14.0	4.0	.....	.....	200-110
April 19	1,300	1,180	1.009	8.8	3.5	3.0	2.7			
April 20	1,300	2,400	1.010	Urea 20 gm. 18.7	8.2	4.0	6.7	.....	.....	210-112
April 21	1,500	1,810	1.010	10.7	8.0	4.0	3.2			
May 3	1,350	1,460	1.008	4.0	4.8	4.0	1.5			
May 4	1,200	1,090	1.010	4.0	4.6	4.0	1.9	.....	.....	170- 98
May 5	1,100	1,355	1.010	4.0	2.8	KCl 5 9.0	7.2	23.7	51	

Case 3 (1154).—This was classified clinically as of chronic nephritis, with albuminuric retinitis and hypertension. Functionally there was a marked inability to excrete added sodium chlorid, with slight inability to excrete added urea. On May 17, 10 gm. of potassium chlorid were added to the diet. No increase of salt excretion occurred. No ill effects were produced. Results are shown in Table 3.

Case 4 (1097).—This was classified clinically as of chronic nephritis with hypertension. Functionally there was a moderate inability to excrete added sodium chlorid, and slight inability to excrete added urea. Ten grams of potassium chlorid were administered on May 8. This was followed by a definite increase in the salt excretion. No ill effects were noted. Results are shown in Table 4.

In two of the cases, additional potassium chlorid produced no increase of chlorid excretion; in two cases there was a definite increase of chlorid excretion. In none of these cases were there any ill effects from the potassium chlorid.

The next case reported was similar both clinically and functionally to those already given.

Case 5 (1158).—A Russian Jewess, aged 42, married, entered the hospital May 5, 1914, complaining of headache and dizziness of a duration of two or three months.

Family History.—The patient's husband and eleven children were living and well. She had had three miscarriages. There was no history of cancer, heart or kidney disease in the family.

Her habit has been to use a moderate amount of tea and coffee for years. Venereal disease is denied.

Past History.—The patient had scarlet fever in childhood; otherwise her general health has been very good. There has been slight dyspnea on exertion for the past few years. Nocturia four or five times for several years has been a troublesome symptom.

Present Illness.—Headaches have been frequent and severe for the past two or three months. Before this time headaches were rare. These headaches are now present two or three days of the week. There has been some dizziness for the past six weeks. A slight puffiness about the face and eyes was noted a month ago. About two weeks ago there was some nausea and vomiting.

Physical Examination.—There is a definite hypertrophy of the heart, the left border being 13 cm. to the left of the midsternum in the sixth space.

A blowing systolic murmur was present, best heard over the sternum. There is no edema of the face or extremities. The fundi of the eyes are normal. Blood pressure, systolic 245, diastolic 120. Physical examination otherwise is negative.

The urine showed a large trace of albumin, with granular casts and many white cells, but no blood. Phthalein test, two hours, 59 per cent.; nonprotein nitrogen, 31.1 mg. per 100 c.c. of blood.

Clinical Diagnosis.—Chronic interstitial nephritis, hypertrophy of the heart, hypertension. Functional tests of the kidney, begun May 16, were not entirely satisfactory because of slight inability to control the urethral sphincter. The added sodium chlorid was poorly excreted; the added nitrogen was excreted fairly well. On May 24, 10 gm. of potassium chlorid were given at 10 a. m. Several hours after taking the salt the patient complained of weakness, abdominal distress and precordial pain. At 5 p. m. she was somewhat cyanotic, markedly prostrated, with regular, rather weak pulse; rate 80. There was considerable abdominal distress and vomiting during the night. At 6 a. m. on

TABLE 3.—Results of Test in Case 3 (1154)

Date	Fluid Intake c.c	Urine		Nitrogen		Chlorid		Mg. N per 100 c.c. Blood	Per Cent. Phthal-ein	Blood Pres-sure
		24° Am. c.c.	Sp. Gr.	Intake Gm.	Output Gm.	Intake Gm.	Output Gm.			
May 8	2,500	2,254	1.010	11.2	13.0	4.0	10.7	30.9	45	
May 9	1,950	1,305	1.013	11.2	9.1	4.0	5.8	.....	.....	220-140
May 10	1,350	1,020	1.021	11.2	10.3	4.0 NaCl 10 gm.	5.3			
May 11	1,500	765	1.025	11.2	6.5	14.0	2.4			
May 12	1,450	940	1.026	9.9	10.9	4.0	3.6	.....	.....	260-160
May 13	1,800	600	1.016	11.2	7.1	4.0	1.1	.....	.....	255-155
May 14	2,700	3,300	1.010	Urea 20 gm. 20.7	19.1	4.0	4.3	.....	60	230-150
May 15	2,100	940	1.020	9.9	9.3	4.0	1.4			
May 16	1,900	1,830	1.017	9.7	9.3	4.0 KCl 10 gm.	0.9			
May 17	2,100	1,150	1.011	8.7	7.3	14.0	2.7	.....	.....	220-150
May 18	1,700	1,330	1.013	20.0	8.8	2.0	4.9			
May 19	1,650	1,245	1.013	22.5	12.0	4.0	2 8	35.1		

TABLE 4.—Results of Test in Case 4 (1097)

Date	Fluid Intake c.c.	Urine		Nitrogen		Chlorid		Mg. N per 100 c.c. Blood	Per Cent. Phthal-ein in 2 Hrs.	Blood Pres-sure
		24° Am. c.c.	Sp. Gr.	Intake Gm.	Output Gm.	Intake Gm.	Output Gm.			
April 30	1,200	1,030	1.019	6.1	8.5	6.0 NaCl 10 gm.	4.0			
May 1	1,500	890	1.022	6.0	7.7	16.0	4.5			
May 2	1,250	550+	1.018	6.0	3.5+	6.0	7.7+	.....	.....	195-110
May 3	1,500	700	1.019	6.0	4.7	6.0	5.5			
May 4	1,200	1,465	1.017	Urea 20 gm. 6.0	12.5	6.0	9.6	.....	.....	180-100
May 5	1,500	410+	1.020	6.0	2.6+	6.0	3.2+	.....	51	
May 6	1,300	Lost	Lost	6.0	Lost	6.0	Lost			
May 7	1,300	840	1.018	11.2	6.4	4.0 KCl 10 gm.	5.2			
May 8	1,500	1,465	1.017	11.2	13.1	14.0	10.2	32.2	.....	190- 85
May 9	1,500	980	1.020	11.2	7.0	4.0	5.8			
May 10	1,200	1,165	1.022	11.2	13.4	4.0	5.2			
May 11	1,550	950	1.022	11.2	10.7	4.0	5.0	.....	51	200-100
May 12	1,250	1,310	1.013	11.2	8.0	4.0	3.7			

the 25th there was a sudden attack of intense cyanosis and marked prostration. There was a diminution in the amount of urine. There had been only a few blood cells in the urine but on the 26th a marked hemoglobinuria appeared. The nonprotein nitrogen in the blood had risen to 84 mg. per 100 c.c. The blood serum showed a definite hemoglobinemia. The spectroscope showed absence of methemoglobin.

TABLE 5.—Results of Test in Case 5 (1158)

Date	Fluid Intake c.c.	Urine		Nitrogen		Chlorid		Mg. N per 100 c.c. Blood	Per Cent. Phthalein 24° 10′	Blood Pressure
		24° Am. c.c.	Sp. Gr.	Intake Gm.	Output Gm.	Intake Gm.	Output Gm.			
May 16	1,495	960	1.016	6.0	6.4	16.0 NaCl 10 gm.	2.2			
May 17	1,500	595	1.015	6.0	4.5	6.0	1.6	.....	.....	180-95
May 18	1,500	895	.....	6.0	.....	6.0				
May 19	1,675	400+	1.014	7.3 Urea 20 gm.	2.6+	4.0	0.76+	.....	52	
May 20	1,280	1,350	1.014	20.0	10.1	4.0	2.5			
May 21	1,330	1,385	1.012	7.0	8.0	3.5	2.3	.....	.....	170-85
May 22	1,310	440+	.....	7.9	.....	3.5				
May 23	590	1,460	1.011	6.3	8.9	3.5	2.2			
May 24	1,635	300+	1.016	5.4	.....	KCl 10 gm.	0.5	.....	.....	184-94
May 25	795	55+	.....	0	?	0	?			
May 26	1,265	408	1.013	0.1	2.5	0	0.4	84.0		
May 27	1,400	310	1.012	0.0	1.9	0	0.3			
May 28	1,350	795	1.012	0.1	5.6	0	0.6	.....	.....	168-85
May 29	1,730	800	1.013	8.0	4.8	3.5	1.4			
May 30	1,760	775+	1.012	5.5	5.3	2.0	0.9	.....	.....	160-75
May 31	1,980	1,280	1.011	5.2	7.0	2.0	1.3			
June 1	1,630	1,200	1.011	8.1	7.2	3.5	1.7			
June 2	1,760	1,275	1.013	7.0	7.2	3.5	1.5			
June 3	1,650	1,700	1.011	8.1	10.9	3.5	1.8			
June 4	1,860	950	1.011	7.5	5.4	3.5	1.1	.....	.....	150-75
June 5	1,910	1,525	1.009	9.8	7.0	4.0	1.5			
June 6	1,810	1,205	1.009	10.5	5.6	4.0	1.2			
June 7	1,560	1,750	1.012	10.0	7.3	4.0	2.1	78.8		
June 20	.....	.....	.....	.....	.....	.....	.....	66.2		
July 1	.....	.....	.....	.....	.....	.....	.....	48.0		
July 4	.....	.....	.....	.....	.....	.....	.....	.....	32	120-58

There was a temperature of 100.8 on the 26th, with an increase in pulse-rate to 98. The temperature and pulse remained elevated for two days.

The symptoms were so severe and resembled so closely those of potassium chlorate poisoning, that at once the question arose as to whether a mistake had been made in the salt given. This was carefully checked and it was soon

proved that the salt given was potassium chlorid. This was substantiated by entire absence of methemoglobin formation. The patient slowly improved and returned to her former condition about June 10. Hemoglobinuria disappeared June 4 and blood cells were gone from the urine June 12. Nonprotein blood nitrogen on the 7th was 78.8 mg., falling to 48.0 on July 4. Throughout her stay in the hospital the blood pressure continued to fall, reaching 120-58 on discharge. She left the hospital with entire relief from headache and dizziness.

This case suggested to us that potassium chlorid in a dose which was harmless in normal individuals might be injurious in nephritis, and since the chlorin ion is devoid of action, that the poisoning must be due either to the action of the potassium ion, or to the "salt action." Since the "salt action" of sodium chlorid and potassium chlorid is the same, and since the patient did not react adversely to 10 gm. sodium chlorid, it seemed probable that the poisoning was due to the potassium ion.

The chief action of potassium in experimental[4] work, is a depression of the heart. There is at first, as a rule, an acceleration of the pulse, then the pulse becomes weaker and slower, and fall in the blood pressure occurs. Bunge[5] has shown that some classes of people—Irish laborers and certain African tribes—have an intake of 50 gm. potassium chlorid a day. The absence of effect on the heart is due to the rapid excretion of the salt by the kidney. Dr. Reid Hunt, in an unpublished experiment, demonstrated that potassium salts in extraordinarily small amounts, produce death in guinea-pigs with the kidneys removed. The guinea-pigs when in normal condition, were injected with various potassium salts without ill effect, for the kidneys excreted the salt so rapidly that the concentration necessary to kill the animal was not reached. With the kidneys removed, much smaller doses of potassium killed the animal at once. Death was probably due to the action of the potassium ion on the heart. He calculated that a man who took a large portion of his food as potatoes, for example an Irish peasant, would have an intake of 10 times the fatal dose of potassium in a day. Were it not for the fact that potassium salts are so rapidly excreted by the kidneys, the effects might be very serious.

Experiments were now made in an attempt to correlate previous laboratory findings and our clinical experience with potassium poisoning in nephritis. Rabbits were given nephritis with uranium nitrate. The degree of nephritis was estimated by frequent blood nitrogen examinations. The same data were kept on rabbits as had been recorded in our patients, with the exception of the specific gravity of the urine, and blood pressure. The rabbits were given a large dose of

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4. Macht: Bull. Johns Hopkins Hosp., 1914, xxv, 278.
5. Bunge: Arch. f. d. ges. Physiol., 1871, iv, 235.

potassium chlorid by mouth while in a normal condition. Uranium nitrate was then given subcutaneously to produce nephritis, and varying doses of potassium chlorid were added to the diet at different periods of the disease. A few typical protocols will be given.

Experiment 1, Rabbit 974.—The animal was given 3 gm. of potassium chlorid in 50 c.c. of water, with rapid excretion of the salt (4 gm. in 50 c.c. of water sometimes caused death in the hot summer). A moderate nephritis was then produced by uranium nitrate. The blood nitrogen rose gradually. The animal was strong and showed no marked symptoms of illness. On the 21st, when the nonprotein blood nitrogen reached 118 mg., one gm. of potassium chlorid was given in 50 c.c. of water. The animal died within fifteen minutes. Necropsy showed typical lesions of acute uranium nephritis (Table 6).

TABLE 6.—Results of Test on Rabbit 974, Experiment 1

Date	Fluid Intake c.c.	Urine c.c.	Nitrogen		Salt		Blood Nitrogen mg. per 100 c.c.	Uranium Nitrate Mg.	Weight Gm.	Remarks
			Intake Gm.	Output Gm.	Intake Gm.	Output Gm.				
Sept. 16	210	150	0.7	1.4	0.15	0.13	.....	....	1,800	
Sept. 17	245	190	0.7	0.78	3.1	2.3	29.2	....	.....	3 gm. KCl
Sept. 18	140	130	0.7	0.59	0.11	0.29	23.7	2		
Sept. 19	200	100	0.7	0.43	0.15	0.12	25.8			
Sept. 20	80	20	0.4	?	0.05	?	52.0			
Sept. 21	50	... Died	.....	.....	1.0	.....	118.0	....	1,840	1 gm. KCl

TABLE 7.—Results of Test on Rabbit 958, Experiment 2

Date	Fluid Intake e.c.	Urine c.c.	Nitrogen		Salt		Blood Nitrogen mg. per 100 c.c.	Uranium Nitrate Mg.	Weight Gm.	Remarks
			Intake Gm.	Output Gm.	Intake Gm.	Output Gm.				
July 20	150	110	0.7	0.55	0.15	0.35				
July 21	200	210	0.7	0.32	3.15	1.50	22.9	....	.....	3 gm. KCl
July 22	200	180	0.7	0.56	0.15	0.52	.....	....	1,850	
July 23	200	60	0.55	0.20	0.15	0.20	.....	2		
July 24	190	154	0.55	0.48	0.15	0.11	38.2			
July 25	200	55	0.30	0.20	0.15	0.09				
July 26	30	48	0.14	0.25	0.04	0.07				
July 27	170	58	0.08	0.15	2.10	0.52	105.6	....	.....	2 gm. NaCl
July 28	140	68	0.40	0.17	0.10	0.39	.....	....	1,800	
July 29	50	... Died	.....	.....	1.0	.....	140.0	....	.....	1 gm. KCl

Experiment 2, Rabbit 958.—The rabbit was given 3 gm. of potassium chlorid, with rapid excretion of the salt. Nephritis was produced with uranium nitrate. When the nonprotein blood nitrogen reached 100 mg., 2 gm. of sodium chlorid

were added to the diet. No ill effects were produced, though there was poor excretion of the salt. Two days later 1 gm. of potassium chlorid was given, with immediate death of the animal. There were no symptoms of weakness nor illness before the potassium chlorid was given. Necropsy showed characteristic lesions of acute nephritis. This experiment suggests that death is not due to "salt action," since the "salt action" of sodium and potassium chlorid is the same.

An attempt was next made to produce a moderate nephritis with repeated injection of uranium, adding to the daily diet small amounts of potassium chlorid.

TABLE 8.—Results of Test on Rabbit 975, Experiment 3

Date	Fluid Intake c.c.	Urine c.c.	Nitrogen		Salt		Blood Nitrogen mg. per 100 c.c.	Uranium Nitrate Mg.	Weight Gm.	Remarks
			Intake Gm.	Output Gm.	Intake Gm.	Output Gm.				
Sept. 16	20	75	0.7	1.1	0.15	0.08	.....	....	2,040	
Sept. 17	150	190	0.1	0.72	4.07	2.8	32.8	....	.....	4 gm. KCl
Sept. 18	120	120	0.7	0.45	0.15	0.32	34.3	2	.....	
Sept. 19	50	38	0.1	0.59	0.02	0.05	33.9			
Sept. 29	...	...	.....	.....	.....	.....	.....	4		
Sept. 30	50	155	0.65	0.56	0.62	0.45	28.4	....	1,920	0.5 gm. KCl
Oct. 1	50	130	0.55	0.48	0.62	0.49	36.7	....	.....	0.5 gm. KCl
Oct. 2	50	105	0.40	0.80	0.63	0.51	53.2	....	1,860	0.5 gm. KCl
Oct. 3	50	150	0.55	.....	0.65	0.69	.....	....	.....	0.5 gm. KCl
Oct. 4	50	85	0.55	0.27	0.65	0.48	60.8	....	.....	0.5 gm. KCl
Oct. 5	50	150	0.70	.....	0.63	0.70	.....	6	1,770	0.5 gm. KCl
Oct. 6	50	150	0.35	0.70	0.60	0.64	58.2	....	.....	0.5 gm. KCl
Oct. 7	50	120	0.3	.....	0.60	0.56	.....	....	.....	0.5 gm. KCl
Oct. 8	50	100	0.55	0.57	0.58	0.45	68.4	....	.....	0.5 gm. KCl
Oct. 9	50	60	0.50	0.14	0.60	0.38	.....	....	.....	0.5 gm. KCl
Oct. 10	50	140	0.60	0.49	0 55	0.82	.....	....	.....	0.5 gm. KCl
Oct. 11	50	140	0.60	0.48	0.60	0.63	54.6	....	1,760	0.5 gm. KCl
Oct. 12	50	180	0.55	.....	1.13	1.20	.....	....	.....	1 gm. KCl
Oct. 13	50	240	0.12 Recovered	.....	1.15	1.1	57.9	....	.....	1 gm. KCl

Experiment 3, Rabbit 975.—Four gm. of potassium chlorid were readily excreted on the 19th. Two mg. of uranium nitrate gave no increase in nonprotein blood nitrogen and the data are omitted. On the 29th four mg. of uranium nitrate were given, with moderate gradual increase of blood nitrogen. Though the nonprotein blood nitrogen rose to 60 mg. per 100 c.c., potassium chlorid was readily excreted and no symptoms were evident. On October 5, 6 mg. of uranium nitrate were given. The nonprotein blood nitrogen rose almost to 70 mg., but the potassium chlorid was well excreted and the animal recovered.

Experiment 4.—This experiment is similar to Experiment 3. Moderate nephritis was produced by repeated injections of uranium nitrate, and potassium chlorid was given at various stages of the disease. The salt was well excreted until the nonprotein blood nitrogen became 100 mg. In this case as in the others, 1 gm. of the salt caused sudden death.

TABLE 9.—Results of Test on Rabbit 978, Experiment 4

Date	Fluid Intake c.c.	Urine c.c.	Nitrogen		Salt		Blood Nitrogen mg. per 100 c.c.	Uranium Nitrate Mg.	Weight Gm.	Remarks
			Intake Gm.	Output Gm.	Intake Gm.	Output Gm.				
Sept. 24	200	80	0.7	0.8	0.15	0.22	.....	....	2,260	
Sept. 25	310	280	0.68	1.0	4.1	3.4	23.8	....	.....	4 gm. KCl
Sept.26	240	180	0.7	0.87	0.15	0.26				
Sept. 27	200	125	0.7	0.86	0.13	0.05	23.1	2		
Sept. 28	245	170	0.7	0.88	0.13	0.12	26.5	....	2,260	
Sept. 29	270	210	0.40	0.60	1.11	1.07	28.2	....	.....	1 gm. KCl
Sept. 30	140	130	0.21	0.54	0.08	0.36	25.4	....	2,230	
Oct. 1	125	130	0.30	0.47	0.07	.....	36.4	3		
Oct. 2	85	90	0.10	0.57	0.03	0.27	36.2	....	2,160	
Oct. 3	120	100	0.50	.....	0.07	0.17	68.0			
Oct. 4	140	85	0.70	0.85	0.06	0.07				
Oct. 5	170	110	0.50	.....	1.1	0.47	57.2	5	2,100	1 gm. KCl
Oct. 6	80	75	0.10	0.42	0.03	0.30				
Oct. 7	100	140	0.10	.....	1.04	0.74	75.6	....	.....	1 gm. KCl
Oct. 8	140	150	0.15	1.0	1.07	0.80				
Oct. 9	180	68	0.10	0.38	0.10	0.14	.....	....	2,040	
Oct. 10	125	180	0.10	1.0	1.07	0.61	93.8	....	.....	1 gm. KCl
Oct. 11	170	180	0.10	0.88	1.08	0.75	.....	....	.....	1 gm. KCl
Oct. 12	50	...	..... Died	.....	1.0	.....	128.6	....	.....	1 gm KCl

Nine other experiments were carried out with similar results. The four examples given are typical. Frothingham, Fitz,[6] and others who have worked with experimental uranium nephritis have shown that death does not occur in the rabbits until the nonprotein blood nitrogen has reached 150, 200, or even 250 mg. per 100 c.c. Furthermore, the animals as a rule show definite symptoms of disease when death is impending.

For our experiments, as is shown, an attempt was made to produce a moderate nephritis, and to give the potassium chlorid before severe symptoms of the disease developed. In each instance, when the non-

6. Frothingham, C., Fitz, R., Folin, Otto, and Denis, W.: The Relation Between Nonprotein Nitrogen Retention and Phenolsulphonephthalein Excretion in Experimental Uranium Nephritis, The Archives Int. Med., 1913, xii, 245.

protein blood nitrogen reached 100 mg. per 100 c.c. the giving of 1 gm. of potassium chlorid caused immediate death. The reason seems obvious, namely, the salt was absorbed by the gastro-intestinal tract, the kidneys were unable to excrete it, and, as in Hunt's experiments, a concentration was reached in the blood which was poisonous to the heart muscle.

Some of the conditions which were present in the experimental animals were also present in the patient, and it is reasonable to assume that some of her symptoms were due to the action of potassium on the heart muscle.

One symptom complex occurred in the patient which was not present in the experimental animals; namely, hemoglobinemia and hemoglobinuria, with rise in temperature and pulse. The cause of this phenomenon will be made the subject of a subsequent paper.

CONCLUSION

1. Rabbits with uranium nephritis of a degree sufficient to increase the nonprotein blood nitrogen to 100 mg. per 100 c.c., die with great suddenness following ingestions of 1 gm. of potassium chlorid.

2. Their death is not due to "salt action," but is probably due to the action of the potassium ion on the heart muscle.

3. In human beings, potassium chlorid, in doses which have no effect on normal individuals, will cause acute poisoning in individuals with chronic nephritis.

4. This acute poisoning occurs because the salt, which is normally readily absorbed and very rapidly excreted, in nephritis is readily absorbed and not excreted, thus reaching a concentration in the blood which is injurious.